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Prenatal Smoking Cessation and Infant Health: Evidence from Sibling Births *

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Abstract

This paper uses a unique large panel data of sibling births to provide new evidence on when prenatal smokers must quit smoking to deliver the healthy newborn. In a series of the mother fixed effect estimation, I find robust results that early cessation in the first trimester nullifies the adverse smoking impact, but late cessation in the second trimester still leaves a remarkably irreversible damage on infant health. About two third of the adverse smoking impact on infant health occurs in the second trimester, mainly through fetal growth retardation. Therefore the first trimester is the critical period for prenatal smoking cessation. In particular, failing to stop smoking promptly in this period is crucial to explain why low socioeconomic status prenatal smokers transmit their poor health and economic status to the offspring. The policy implication is that reallocating resources on prenatal smoking cessation towards the first trimester can lead to a significant efficiency gain. This paper also uncovers a new source of downward bias in estimating the causal relation between a group measure "prenatal smokers" and infant health, if the timing information of smoking cessation is misused.

Keywords: Prenatal Smoking, Timing of Smoking Cessation, Birth Weight, Low Birth Weight

JEL Numbers: I12, I18.

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1. INTRODUCTION

It has been well established that prenatal smoking can substantially lower infant birth weight, increase the risk of delivering low birth weight(LBW)[1] babies, and cause a host of other adverse birth outcomes (CDC, 2001). Adams *et al.* (2002) reports the short term neonatal costs attributable to prenatal smoking is over $700 per pregnant smoker(in 1996 dollars). More important, poor infant health due to prenatal smoking often results in childhood developmental problems, low educational attainments(Hack *et al.*, 1995; Lewit *et al.*, 1995; Corman and Chaikind, 1998) and adverse labor market outcomes (Case *et al.*, 2005). Because of the large intergenerational health and economic costs of prenatal smoking, promoting smoking cessation among pregnant women is a current focus of policy makers and medical practitioners. However one important question on such intervention[2] remains unsettled: at which stage during pregnancy is smoking cessation absolutely necessary for the mothers to nullify the adverse smoking impact on infant health? The literature has suggested a fast accumulation of the adverse prenatal smoking impact on babies primarily occurs either at the beginning of the second or that of the third trimester. Therefore the question is narrowed down to whether smoking cessation as late as in the second trimester(late cessation)[3] still leaves a remarkably irreversible damage on infant health. If so, the first trimester is when any kind of prenatal smoking cessation treatment must concentrate on(early cessation). Otherwise late cessation should be underscored. In this case a prolonged and increasingly extensive cessation intervention especially within the second trimester can be more cost effective[4].

The past studies on the timing of fetal exposure to prenatal smoking have nonetheless universally used cross-sectional non-experimental data, and rarely taken into account the

[1]The weight of live born infants less than 2500 grams.

[2]Pregnant women are usually asked about their smoking status at the first prenatal visit. Then the physicians usually advise each smoker to carefully think of quitting, provide pregnancy-specific smoking cessation materials and develop a specific quit plan.

[3]Below I define the prenatal smokers who quit smoking in the first trimester as "early quitters", and those who quit smoking in the second trimester as "late quitters". The data used in this paper codes whether a mother has smoked in any trimester or three months prior to pregnancy. So the category called "continuous smokers" includes the mothers who either never quitted smoking during pregnancy or quitted smoking in the third trimester (The third trimester quitters account for a tiny fraction of prenatal smokers by the literature).

[4]Heavy prenatal smokers usually need more time to get rid of the dependence on cigarette. If late cessation can nullify the smoking impact, then such cessation counseling can achieve a higher quitting rate by the end of the second trimester and protect more infants from the irreversible harm of prenatal smoking.

endogeneity of smoking cessation. Then an immediate concern is whether the unobserved mother heterogeneity can drive the previous controversial findings. For example, mothers who delay smoking cessation during pregnancy are probably involved in heavy alcohol drinking, illicit drug use and other complementary unhealthy activities most of which are often unobserved. Consequently the impact of late cessation is overstated, making the first trimester more like the critical smoking cessation period. However, the disadvantaged mothers with low health endowment can compensate for the newborn by quitting much earlier and using health augmenting inputs[5], biasing downward the estimate on late cessation to zero. It then suggests that the pregnant women can nullify all the adverse smoking impact even they delay cessation until the second trimester. This paper makes the first attempt to eliminate the common maternal influence on both smoking cessation and birth outcomes.

Understanding the relation between prenatal smoking cessation and infant health is crucial to improve the cost effectiveness of any smoking cessation intervention for childbearing women. If early rather than late cessation is absolutely necessary for nullifying the negative smoking impact on the birth outcomes, reallocating resources on smoking cessation towards the first trimester can lead to a significant efficiency gain. This study is also important for the researchers to correctly define "prenatal smokers", when the timing information of prenatal smoking cessation is available. For instance, although prenatal smoking has been considered as a key input for the newborn's health in the large literature of infant health production (Rosenzweig and Schultz, 1983; Corman *et al.*, 1987; Reichman *et al.*, 2009; Noonan *et al.*, 2007), little attention is paid to the timing issue of smoking cessation. Nonetheless, if the early quitters can nullify the adverse smoking impact while the late quitters cannot, then the well-defined "prenatal smokers" should only include those who kept on smoking through the second or third trimester.

This paper makes three important improvements on the past research. First, it uses a panel data of sibling births to about 80000 mothers living in Pennsylvania or Washington. This is the largest sample ever used to address the link between smoking cessation and infant health. The data is based on the universe of births in the two states and has a rich set of birth outcomes, mother smoking cessation and parental demographics, etc. Hence

[5]In other words, the mothers who can handle the adverse smoking impact are more likely to smoke through the second trimester.

it is superior to other datasets used before which are often convenience samples or contain very limited socioeconomic information. Second, this paper applies a mother fixed effects approach to handle the endogeneity of prenatal smoking cessation. The baseline specification also controls for the existence of any salient medical risk factor which can correlate with smoking cessation and infant health. Third, this study uncovers a new source of potential bias in estimating the causal effect of prenatal smoking, when the timing of smoking cessation is incorrectly used to define a group measure "prenatal smokers". The literature so far has addressed two other cases: changes in the average intake of harmful substances per cigarette among prenatal smokers (Fertig, 2010[6]) and prenatal smokers non-randomly misclassified as nonsmokers (Brachet, 2005).

The rest of the paper proceeds as follows. Section 2 reviews the literature. Section 3 describes the data and the empirical methods. Section 4 reports results, conducts robustness checks and discusses the findings. Section 5 concludes.

2. LITERATURE

There are many epidemiological studies on how the timing of prenatal smoking cessation affects infant health, yet the evidence is mixed. The controversy centers upon whether a fast accumulation of the negative smoking effect occurs either at the beginning of the second or that of the third trimester, or whether late smoking cessation still leads to a noticeable poor birth outcome. The optimistic findings in Rush and Cassano (1983) and Lindley *et al.* (2000) suggest that the smoking cessation deadline can be as late as the first month of the third trimester. Similarly, Lieberman *et al.* (1994) shows that if mothers can quit smoking by the end of the second trimester, the risk of delivering undersized infants will be equal to nonsmokers. In contrast, Macarthur and Knox (1988) reports smoking cessation after the 16^{th} week which is the beginning of the second trimester can only mitigate but not nullify the smoking impact on babies. A lot of recent studies (McDonald *et al.*, 1992; Wisborg *et al.*, 2001; Wang *et al.*, 2002) further indicate that it is necessary for smoking mothers to quit before the second trimester to make their infants as healthy as the nonsmokers. Several limitations are salient in this literature. First, the samples are usually highly selective from

[6]See Adda and Cornaglia (2006) for this behavior among the general smoking population.

a few cities or hospitals, or lack important information on the mothers or families. Second, only cross-sectional non-experimental analyses are provided.

Three general methods have been frequently applied to deal with the unobserved mother's heterogeneity in prenatal smoking, especially in the economics literature. The first is ordinary least square (OLS) or matching estimation when a rich set of covariates are available such that the typically unobservable factors correlated with both prenatal smoking and infant health can be controlled for(Almond *et al.*, 2005; Reichman *et al.*, 2009). The second approach is to instrument for prenatal smoking. The instrumental variables include a random assignment of a smoking cessation intervention (Permutt and Hebel, 1989; Sexton and Hebel, 1984), state cigarette tax rates (Evans and Ringel, 1999) and state cigarette tax hikes (Lien and Evans, 2005). The third is to use panel data. The early works by Rosenzweig and Wolpin (1991, 1995) examine National Longitudinal Survey of Youth 1979, while Abrevaya (2006) works on a large matched panel data of sibling births[7]. All of them utilize a mother fixed effects model and show the smoking impact on infant health is remarkably smaller than those found by the previous two strategies. Abrevaya and Dahl (2008) recently applies a panel quantile estimation to find the smoking effect ranges from -80 to -20 grams (g) between the 20% and 80% quantiles. None of the studies has yet considered the timing of smoking cessation[8]. Consequently the mothers smoking in any trimester were defined as "prenatal smokers". This definition is problematic if the early cessation can nullify the adverse smoking impact while late cessation cannot. Below this paper shows the estimates on such "prenatal smokers" are subject to a downward bias using the available information on the timing of smoking cessation. Similarly, treating the late quitters as "prenatal nonsmokers" can introduce another downward bias.

This work merges the above two strands of literature. It uses a unique large panel data of sibling births to examine the relation between prenatal smoking cessation and birth outcomes. A mother fixed effects model is applied to handle the endogeneity of prenatal smoking cessation. I also show why the timing of smoking cessation matters in estimating

[7]Lack of mother-specific identifiers is a concern. But that study uses a proxy to gauge the degree of correct matching and thus provides estimates on more precisely matched birth pairs. The connection between those estimates and my results is discussed below.

[8]This is mainly because none of the surveys or administrative data publicly available to the researchers had coded smoking at different stages of pregnancy until very recently.

the causal link between a single measure "prenatal smokers" and infant health.

3. DATA AND EMPIRICAL METHODS

The panel data for this study consists of two components: a subsample of Washington State Longitudinal Births Database 2007 (WSLBD07) and a matched panel of Pennsylvania State sibling births. They are constructed from the Natality data of the universe of births in each state and contain all the mothers with two or three singleton births[9] between 2003 and 2006[10]. In the appendix, I illustrate the detail data construction procedure. In particular, it describes how the restricted information such as mother's name, mother's date of birth and infant's date of birth has been used with other demographic variables to precisely match sibling births to the same mother. This sample is suitable to address the present research question for three reasons. First, its panel structure can be used to handle the mother's unobserved heterogeneity. Second, it has a rich set of mother and family level controls. Third, in each state there was a large population of childbearing women, among whom prenatal smoking had been prevalent in the sample period (Appendix Table AI).

The data code each mother's number of cigarettes smoked per day at three months before pregnancy, the first, second and third trimester. The response rate to the smoking questions is very high, 97.55 percent for Washington mothers and 98 percent for Pennsylvania mothers. Although there are 16 combinations of smoking at each of the aforementioned four stages, only five are dominant cases because mothers tend to smoke continuously rather than intermittently before quitting (Column 7 to 10, Appendix Table II). I thus create four mutually exclusive smoker categories for the five cases, with the nonsmokers as the base.They are called "cessation before pregnancy"(Sk_0),"cessation in the 1^{st} trimester"(Sk_1),"cessation in the 2^{nd} trimester"(Sk_2)[11], and "smoking throughout"(Sk_3). The excluded intermittent smokers account for less than 1 percent of all the mothers or 5 percent of all the smokers.

[9]Only a very few number of mothers gave birth to more than three babies in the sample period. I exclude them from the sample.

[10]This period is the focus because 2003 is the beginning year for Washington and Pennsylvania to code smoking cessation at different pregnancy stages (some other states started later on, see Appendix Table AI), while the most recent confidential data that can be released is 2006 when I launched this research project in 2009.

[11]Note mothers in the category of "cessation in the 1^{st} trimester" ("cessation in the 2^{nd} trimester") are "early quitters"("late quitters").

Below I extend the analysis to the more broadly defined smokers in which the mothers smoking continuously or intermittently until the same stage are grouped together (Column 5 and 6, Appendix Table II). In order to make a clear distinction between the late quitters and continuous smokers, I focus on the mothers with at least 30 gestation weeks in the backbone analysis. Only 0.83 percent of all the matched mothers are then dropped. With this sample restriction, the fetuses of all the late quitters were free from exposure to smoking for at least three weeks fewer than the continuous smokers[12]. Later I also examine the mothers with at least 28 gestation weeks. The results are similar.

The descriptive statistics for the siblings data are presented in Table I. The birth outcomes of interest are birth weight and LBW. Birth weight is the primary and most frequently addressed measure of infant health. LBW is a key indicator of poor health at birth, which has a lasting adverse impact on one's health, cognitive development, earnings, and other lifetime outcomes. Prenatal smoking can increase the risk of LBW by either slowing down fetal growth rate or shortening gestation. While fetal growth retardation due to smoking has received much attention in the literature, whether and why prenatal smoking leads to premature births has not been well understood (Kramer, 1987). Hence, below I also address the link between smoking cessation and fetal growth rate by controlling for gestation. Many infant, maternal or paternal control variables are used in this study. I follow the standard approach by coding missing values in separate indicators. The two-birth mothers delivered heavier infants than those with three births, and they were more likely to be nonsmokers, better educated and married.This pattern is not driven by the short four-year sample period because it is consistent with Royer (2004) which uses a panel of mothers who had given birth over 12 years[13].

[Insert Table I]

Table II compares the nonsmokers and the four different types of smokers using the cross sectional birth observations. The late quitters and continuous smokers were similar in most of the observed characteristics. Yet both groups were more likely to be unmarried, receive

[12]The third trimester starts at the 27^{th} week.

[13]Moreover, there are fewer missing values on father's age or education thus fewer unplanned pregnancies (Waston and Fertig, 2009) among the two-birth mothers. It suggests that the unintended pregnancies are less likely to drive both prenatal smoking cessation and infant health for this group of mothers.

less education and have unplanned pregnancies, in contrast to the nonsmokers and early quitters. A higher proportion of the mothers who smoked through the second trimester were also enrolled in the Medicaid or the Women, Infants and Children program (WIC). Therefore a mother's low socioeconomic status (SES) is associated with delayed prenatal smoking cessation. It is well known a poor in-utero environment due to prenatal smoking is important to explain why the low SES mothers transmit their health and economic status to the next generation. The present study stresses at the heart of this mechanism is failing to quit smoking timely during pregnancy, because it shows below that late cessation results in a salient loss in infant health which in turn negatively affects many lifetime outcomes. Taking into consideration the timing of smoking cessation, this paper provides a new insight on how exposure to smoking in-utero affects the intergenerational transfer of health and wealth among the poor[14].

[Insert Table II]

To assess the relation between prenatal smoking cessation and infant health, I begin with the following reduced form model on infant health production:

$$Y_{ij} = \alpha + \sum_{m=0}^{3} \beta_m Sk_{ijm} + \sum_{n=0}^{N_1} \gamma_n X_{ijn} + \varepsilon_{ij} \tag{1}$$

where Y_{ij} is a health measure such as birth weight or LBW of an infant of birth order j to mother i. SK is a vector of four smoker indictors as mentioned above. The X is a rich set of control variables, including birth characteristics (infant male, parity, birth year/month), parental demographics (age, race/ethnicity[15], and education), mother socioeconomic background, prenatal care (Kessner index[16]), indictors for delivery payment types such as Medicaid, participation in WIC, number of other pregnant outcomes, any medical risk factor[17],

[14] See Currie (2009) for a recent summary.

[15] The original Natality Birth data separately code every mother's Hispanic origin and race. I first create two race indicators for the mothers with no Hispanic origin, and then construct another indicator for the Hispanic mothers. The same procedure applies to the fathers. A few Washington mothers have reported multiple races. Thus their race indictors are not mutually exclusive.

[16] Kessner index is a standard measure on the adequacy of prenatal care. It can take values of 1(adequate), 2(intermediate) or 3(inadequate).

[17] The Pennsylvania data code nine risk factors in total: pre-pregnancy diabetes, gestational diabetes, pre-pregnancy hypertension, gestational hypertension, previous pre-term birth, previous poor pregnancy outcomes, vaginal bleeding, and pregnancy resulted from infertility treatment and previous cesarean. The Washington data code one more risk factor: group B streptococcus culture positive.

etc. The results below are almost the same when the model specification includes birth order effects instead of parity. Besides, ε_{ij} is an infant specific component of health.

However, the estimates on β in Equation(1) are biased when the unobserved mother heterogeneity is associated with smoking cessation and infant health. If this heterogeneity is birth invariant and mothers do not respond to the infant specific health shock of the previous birth, then estimating the following mother fixed effect model gives the consistent estimates on β:

$$Y_{ij} = \alpha + \sum_{m=0}^{3} \beta_m Sk_{ijm} + \sum_{n=0}^{N_2} \gamma_n X_{ijn} + \mu_i + \varepsilon_{ij} \tag{2}$$

where μ_i is the mother fixed effect and $N_1 > N_2$. It can capture the time-invariant unobserved health augment characteristics of a mother such as her taste for healthy behavior, her health endowment (Rosenzweig and Wolpin, 1991, 1995), and her earnings potential. The direction and magnitude of the biases in estimating each β_i by Equation(1) depends on the correlation between μ_i and SK_m. For instance, the late quitters or continuous smokers are more likely to heavily use other harmful substances (alcohol, illicit drug, etc.) or have poor nutrition intake, all of which are typically unobserved. In this case, SK_2 and SK_3 are strongly negative correlated with μ_i[18], thus sizably biasing upward the estimates on β_2 and β_3. Table II provides some evidence for this story. There is a noticeable negative selection to the late quitters and continuous smokers in education, marital status, unplanned pregnancy and other observed characteristics. Hence the same selection pattern may also hold for the unobservable. As the other possibility, a mother's prenatal behavior can compensate for the cross generation transmission of her genetic disadvantage, poor health or wealth. The mothers with low health endowment who would have smoked through the second trimester choose to quit promptly prior to pregnancy and allocate other healthy inputs. Although less plausible from Table II[19], this story means that SK_2 and $SK3$ are strongly positive correlated with μ_i[20], which leads to a large downward bias toward zero on the estimates of SK_2 and $SK3$ in Equation(1). A comparison on the results from the two specifications will

[18]The correlation between SK_0 or SK_1 and μ_i is probably weaker. The earlier quitters are less addicted to cigarette; so their dependence on other complementary substances may be lower as well.

[19]It shows the low SES mothers tend to postpone rather than expedite smoking cessation.

[20]Mothers who would have smoked only in the first trimester may not have such a strong compensatory incentive to actually quit much earlier before the pregnancy, since they know the short term prenatal smoking does little harm on the newborn. So the correlation between Sk_1 and mu_i is again possibly weaker. The same for Sk_0.

indicate whether mothers with low μ exacerbate or mitigate the smoking impact especially for the late quitters and continuous smokers.

4. RESULTS

Figure 1 represents the primary relationship between smoking cessation and infant health for the one- to three-birth mothers whose gestation weeks are at least 30[21]. The top bar graph shows conditional on ever smoking mothers, the smoking cessation patterns over different stages prior to and during pregnancy were very similar in both states except that a higher fraction of mothers smoked in Pennsylvania. About 20 percent of the smoking mothers quitted three months before pregnancy. For those who kept on smoking, 10 to 15 percent quitted in the first trimester, about 5 percent in the second trimester[22], others throughout the three trimesters. The middle graph indicates the average infant birth weights for the mothers who quitted smoking prior to pregnancy or in the first trimester were very close to nonsmokers. However, the babies of the late quitters were much lighter (about 120 to 130 g lighter than the early quitters). There is only a moderate decline on infant birth weight if mothers kept on smoking through the third trimester. The bottom graph shows similar LBW rates across three groups of mothers who did not smoked, quitted smoking prior to pregnancy or in the first trimester, but then a remarkably 3.4 to 3.9 percentage points increase in LBW for the late quitters. Figure 2 indicates the pattern on smoking cessation and infant health is virtually the same as Figure 1 for the sibling birth sample which this paper focuses on. It is also robust to the mothers with at least 28 gestation weeks (Appendix Figure AI) or more broadly defined smokers which include the intermittent smokers[23].

[Insert Figure 1]

[Insert Figure 2]

Table III presents the baseline results for the two-birth mothers with at least 30 gestation weeks. Column 1 shows the estimates of Equation(1) for the Washington sample. The adverse effects of smoking cessation prior to pregnancy or in the first trimester on birth weight (Upper Panel) and LBW (Lower Panel) are small and statistically insignificant. But late

[21]They represent over 97 percent of the births given in the sample period.
[22]It corresponds to about 5500 late quitter-birth pairs in the two states.
[23]The figure is available upon request.

cessation leads to much worse birth outcomes, reducing birth weight by 100 g and increasing LBW by 4 percentage points. The continuous smokers have even lighter babies. Column 2 controls for the mother fixed effects, shrinking the estimated impact on late cessation to -64 g on birth weight and an insignificant 3 percentage points increase on LBW. The estimates on continuous smokers are also sizably reduced. A comparison of such two columns suggests that mothers with low health endowment tend to exacerbate rather than compensate for the impact of late cessation or continuous smoking, consistent with Rosenzweig and Wolpin (1995), Abrevaya (2006), and Abrevaya and Dahl (2008). Column 3 and 4 also shows the early rather than late cessation can nullify the negative smoking effect on the Pennsylvania sibling births. In contrast to Column 1, the Pennsylvania early quitters are more negatively selected such that there is a significant yet small impact of early cessation on birth weight in Column 3 (a tiny bias for the one on LBW). But it is eliminated as the mother fixed effects are added in Column 4. The estimates on the pooled sample are represented in the last two columns. Column 6 shows late cessation leads to a decline of 68 g on birth weight and an increase of 1.2 percentage points on LBW. Two third of the negative smoking impact on infant health occurs in the second trimester.

[Insert Table III]

Table IV provide the evidence on a larger sample of two- or three-birth mothers. The results are very similar to Table III. Column 6 shows for the pooled sample early cessation nullify the negative smoking impact, but late cessation reduces birth weight by 77 g and increases LBW by an insignificant 1.4 percentage points. Table V present the results for two-birth mothers whose gestation weeks were at least 28. As mentioned above, the previous sample restriction of 30 gestation weeks can clearly distinguish the late quitters from the continuous smokers. Yet if the second trimester smoking also decreases gestation, this restriction can bias downward the estimate on late cessation. Relaxing it to 28 weeks[24] reduces this bias. Indeed, the estimates on late cessation in Table V are now uniformly larger[25] than the ones in Table III. Column 6 shows for the pooled sample prenatal smoking does no

[24]Using a shorter gestation is problematic because it increases the chance of reverse causality. For instance, mothers with 27 of gestation who self-reported as late quitters may actually quit at the end of the second trimester right after delivering an unhealthy baby.

[25]This is because the fetuses of late quitters in this sample had on average shorter periods being free from smoking exposure.

harm on infant health with early cessation, while late cessation leads to a decline of 97 g on birth weight and an increase of 2.5 percentage points on LBW once the mother fixed effects are controlled for. Note both of the estimates are statistically significant.

[Insert Table IV]

[Insert Table V]

Table VI summarizes the results when gestation is controlled. The smoking estimates now capture how different cessation statuses affect the fetal growth rate. Again we focus on the results by the mother fixed effects estimation on the pooled sample. Column 6 shows that late cessation is associated with a 56 g decrease on birth weight for gestation and an insignificant 1 percentage point increase on LBW for gestation. Comparing the results with Column 6 of Table V, I find that about 60 percent of the late cessation impact on birth weight operates through fetal growth retardation. Besides, I also examine the sample of one-birth mothers to assess the generalizability of the baseline results from the sibling births. The results not reported here are very similar to Table III.

[Insert Table VI]

Table VII first applies the baseline analysis to the more generally defined smokers and then considers the role of smoking intensity. With the definition extending over the intermittent smokers, two third of the newly included continuous smokers did not smoke in the second trimester while all of the newly added late quitters did smoke in the second trimester. The percentage increase in the number of continuous smokers and late quitters are also similar (7 percent). If a large fraction of the adverse smoking impact has accrued since the beginning of the second trimester, the new estimate on late cessation should be closer to the one on smoking throughout. Column 2 shows as expected the difference of the two birth weight estimates on late quitters and continuous smokers is 13 g, smaller than the previous 22 g (Column 6, Table III) after eliminating the constant maternal influence. Column 6 indicates the gap of the two corresponding estimated impacts on LBW shrinks to 0.1 percentage point while it is 0.6 percentage point by Column 6 of Table III. Column 2 and 6 show that late cessation is significantly associated with a 72 g decrease on birth weight and a 1.8 percentage point increase on LBW. Intensive cigarette consumption can worsen birth outcomes of the smokers, conditional on their cessation decisions. Column 4 indicates smoking more than 10

cigarettes per day in the first and second trimester leads to an extra 20 g birth weight loss for the babies of continuous smokers, while the estimate on late quitters is insignificant. Since there are more heavy smokers in the Pennsylvania sample, this finding can partly explain why the late quitters and continuous smokers in this state tend to have lighter babies than Washington[26]. Column 7 and 8 show the link between heavy smoking and any further increase in LBW is not statistically significant.

[Insert Table VII]

Table VIII highlights how much treating the late quitters as "prenatal nonsmokers" or the early quitters as "prenatal smokers" can bias the estimate on the group measure of "prenatal smokers"[27]. The benchmark comparison case is that the "prenatal smokers" only consist of the late quitters and continuous smokers. I focus on the pooled two-birth mothers sample without any restriction on gestation. Column (1) shows the estimates are similar across the three definitions of "prenatal smokers" without controlling for the mother fixed effects. Column (2) however gives quite a different result when the mother fixed effects are added. On one hand, coding the late quitters as "prenatal nonsmokers" such that only the continuous smokers (smoking in ever trimester) are defined as "prenatal smokers" biases downward the estimated impact on birth weight and LBW by a third and over a half respectively[28]. On the other hand, regarding the early quitters the same as the late quitters or continuous smokers gives the estimated smoking impact of -71 g on birth weight and a 2 percentage points on LBW. Interestingly, they are close to Abrevaya (2006) which uses the same smoker definition and a similar matched Natality Data of earlier years. It shows the fixed effects estimates are -79 to -67 g on birth weight and 1 to 1.3 percentage points on LBW when he uses the sample of the most precisely matched birth pairs between 1990 and 1994. Table VIII also shows coding the mothers smoking in any trimester as "prenatal smokers" biases downward the impact estimate on birth weight by about a quarter yet the one on LBW is small.

[Insert Table VIII]

[26]See Column 2 and 4 in Table III, IV and V. This difference is noticeable for the estimated effects on birth weight. The contrast on LBW is less clear. However note the estimates on LBW are often associated with large standard errors.

[27]To simplify the analysis, I do not consider other types of measurement errors on prenatal smokers.

[28]The ratios are calculated by comparing Row 3 and 7 with Row 1 of Column (2).

My findings corroborate two points in Fertig (2010) which uses three British cohorts (1958, 1970 and 2000) to examine how much the association between prenatal smoking and infant health is due to selection. First, she codes mothers in the 1958 and 2000 cohorts who reported smoking after the fourth month of pregnancy as "prenatal smokers". This definition is appropriate since it is consistent with the benchmark case in Table VIII. Second, because the timing of prenatal smoking cessation was not coded in the 1970 cohort, she has to treat all the late quitters as "prenatal nonsmokers". The estimated selection into smoking effect in 1970 is smaller than 2000, consistent with the original hypothesis that mothers were gradually aware of the smoking hazards. However, Fertig (2010) points out that another contributor to this finding may be the grouping of the late quitters with nonsmokers if late cessation leads to poor birth outcomes. Table VIII shows indeed this possibility cannot be ruled out.

5. CONCLUSION

As a key modifiable risk factor for poor infant health, prenatal smoking generates substantial intergenerational costs on health care, education and public assistance systems. This paper uses a unique large panel data of sibling births to provide new evidence on when mothers must quit smoking during pregnancy to keep the newborn healthy. I find early cessation in the first trimester nullifies the adverse smoking impact. However, late cessation in the second trimester still leaves a noticeable damage on infant health. The baseline mother fixed effects impact estimates of late cessation are -68 g on birth weight and 1.2 percentage points on LBW (a 30% increase), controlling for the presence of any salient medical risk factor. The corresponding estimates of smoking throughout are -90 g and 2 percentage points. About two third of the adverse smoking impact on infant health occurs in the second trimester, mainly through fetal growth retardation. The relationship between smoking cessation and birth outcomes remains robust to mothers with two or three births, or those with at least 28 gestation weeks, or a set of more broadly defined smoker indicators which considers the intermittent smokers. I also find either treating the late quitters as "prenatal nonsmokers" or the early quitters as "prenatal smokers" can introduce a nontrivial downward bias in estimating the causal relation between the group measure "prenatal smokers" and infant

health.

This paper reaches four conclusions. First, there is a rapid accumulation of smoking impact on infant health in the second trimester. Therefore the first trimester is the critical period for prenatal smoking cessation. Second, researchers should focus on both the late quitters and continuous smokers as "prenatal smokers" when the timing information of prenatal smoking cessation is available. Third, mothers with low health endowment tend to sizably exacerbate rather than compensate for the harmful impact of late smoking cessation or smoking throughout. Fourth, failing to stop smoking promptly during pregnancy plays a key role in explaining why low SES prenatal smokers transmit their health and economic status to the offspring.

The policy and practical implication of this paper is straightforward. Prenatal smoking cessation intervention must concentrate on the first trimester (early cessation) when pregnant smokers are advised to carefully consider quitting at the initial prenatal visit. This new guideline is critical to improve the efficacy of any type of cessation counseling or insurance program (for example, Medicaid) which covers the prenatal smokers' cessation expense. In evaluating the cost-effectiveness of a specific cessation treatment such as 5 A's[29], we should not only look at the quitting rate but also examine how successfully it can achieve early cessation among prenatal smokers.

[29]From 2000 to 2005, the Robert Wood Johnson Foundation (RWJF) provided grant supports for the American College of Obstetricians and Gynecologists (ACOG) to promote a five step smoking cessation guideline as a routine part of prenatal care for pregnant women. This intervention is known as the 5 A's (Ask, Advise, Assess, Assist, Arrange).

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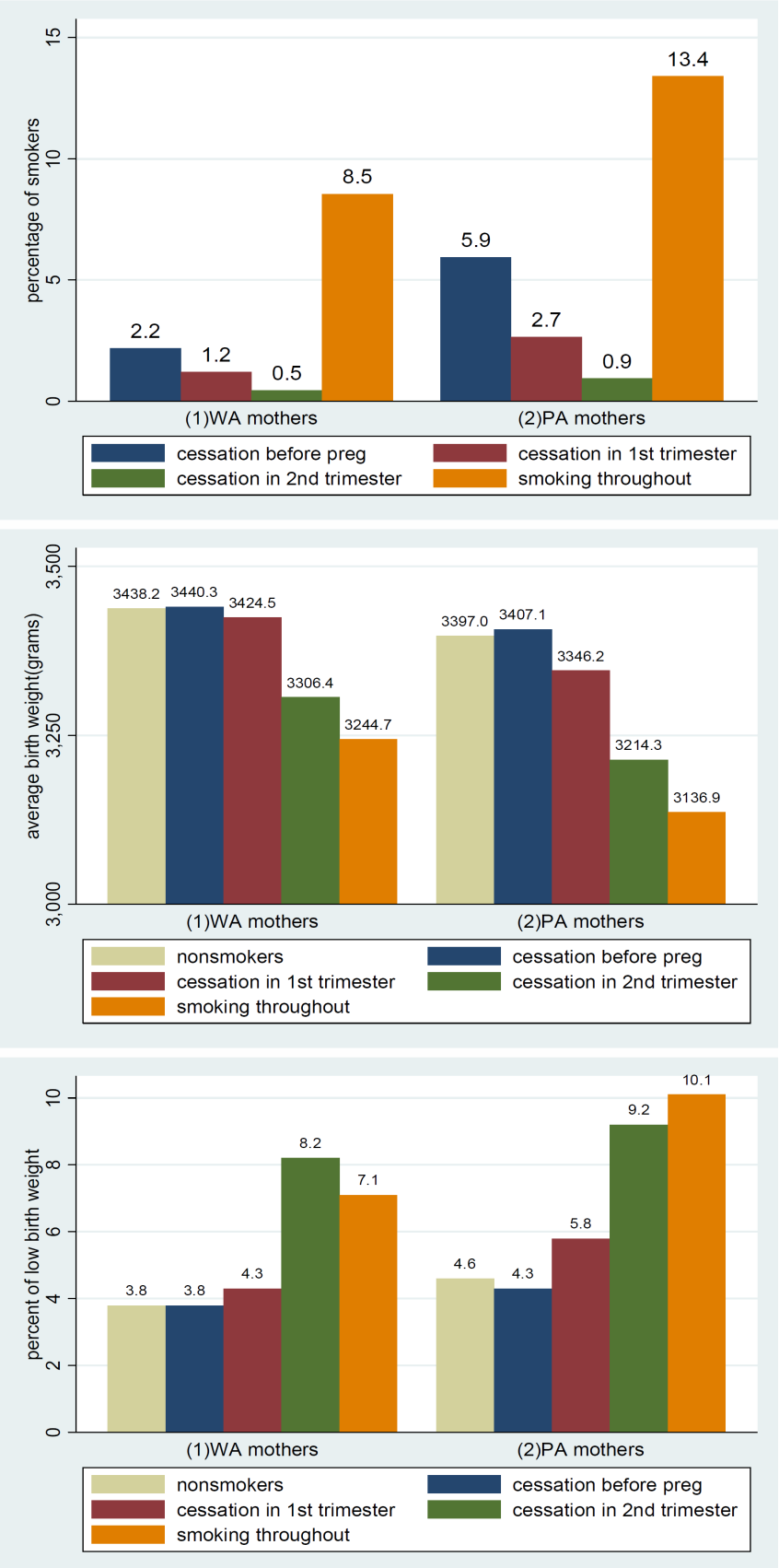

Figure 1: Smoking Cessation and Infant Health (Mothers with 1 to 3 Births, Gestation ≥ 30)

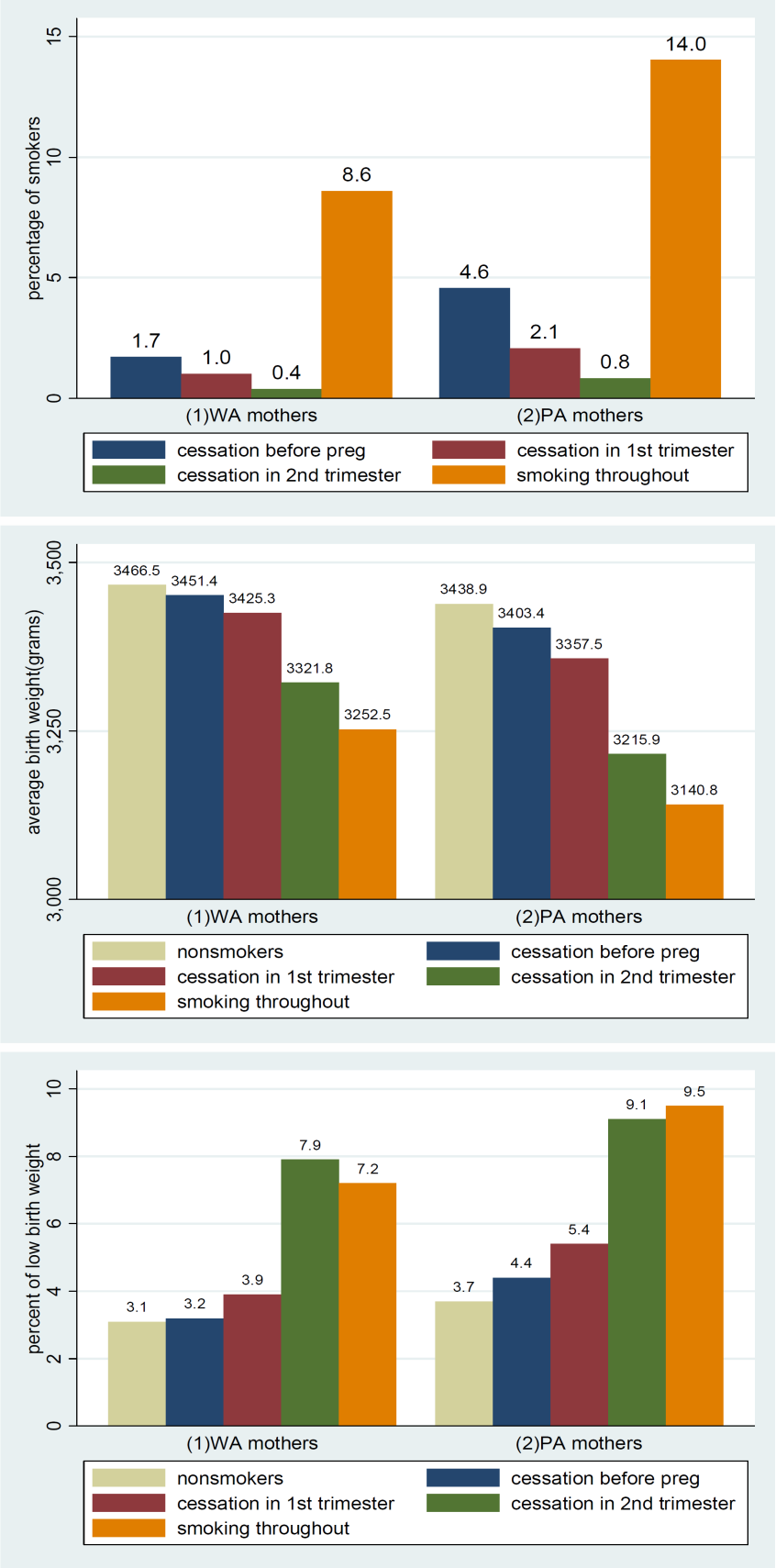

Figure 2: Smoking Cessation and Infant Health (Mothers with 2 or 3 Births, Gestation ≥ 30)

Table I: Descriptive Statistics(Gestation ≥ 30)

Variable	Mothers at WA				Mothers at PA			
	2 births in 03-06		3 births in 03-06		2 births in 03-06		3 births in 03-06	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Birth Weight(grams)	3448.021	510.252	3424.959	538.367	3393.420	523.556	3370.182	531.685
Low Birth Weight	0.034	0.182	0.043	0.203	0.046	0.209	0.049	0.215
Gestation (weeks)	38.903	1.508	38.775	1.650	38.854	1.577	38.840	1.651
Cessation before Pregnancy	0.017	0.131	0.014	0.115	0.047	0.211	0.032	0.176
Cessation in the 1^{st} Tri	0.010	0.100	0.011	0.105	0.021	0.144	0.017	0.129
Cessation in the 2^{nd} Tri	0.004	0.061	0.006	0.075	0.008	0.091	0.008	0.088
Smoke throughout	0.084	0.277	0.131	0.338	0.137	0.344	0.183	0.387
Infant Male	0.515	0.500	0.512	0.500	0.513	0.500	0.511	0.500
Parity	1.265	1.356	2.084	1.934	1.290	1.427	2.051	1.786
Mother's Age	27.492	5.690	25.655	5.428	27.403	5.576	25.607	5.183
Mother non Hisp White	0.737	0.440	0.724	0.447	0.847	0.360	0.810	0.392
Mother non Hisp Black	0.033	0.178	0.044	0.206	0.109	0.311	0.145	0.353
Mother Asian	0.068	0.251	0.044	0.205	0.011	0.104	0.007	0.085
Mother Hispanic	0.026	0.160	0.036	0.186	0.031	0.173	0.035	0.183
Mother Edu = 12 yrs	0.233	0.423	0.308	0.462	0.266	0.442	0.270	0.444
Mother Edu 13-15 yrs	0.297	0.457	0.279	0.449	0.244	0.429	0.176	0.381
Mother Edu $\geq$ 16 yrs	0.286	0.452	0.144	0.351	0.312	0.463	0.179	0.384
Mother Edu missing	0.011	0.104	0.010	0.100	0.003	0.056	0.004	0.065
Mother Married	0.753	0.431	0.692	0.462	0.705	0.456	0.629	0.483
Father's Age	30.250	6.127	29.029	6.064	30.094	6.014	28.878	6.008
Father's Age missing	0.069	0.253	0.109	0.311	0.064	0.245	0.100	0.301
Father non Hisp White	0.676	0.468	0.630	0.483	0.783	0.412	0.717	0.451
Father non Hisp Black	0.036	0.187	0.054	0.226	0.114	0.318	0.151	0.358
Father Asian	0.055	0.228	0.034	0.180	0.009	0.097	0.007	0.084
Father Hispanic	0.237	0.425	0.260	0.439	0.082	0.274	0.114	0.318
Father Race missing	0.093	0.291	0.135	0.341	0.041	0.199	0.058	0.234

Table I(Continued): Descriptive Statistics(Gestation ≥ 30)

Variable	Mothers at WA				Mothers at PA			
	2 births in 03-06		3 births in 03-06		2 births in 03-06		3 births in 03-06	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Father Edu = 12 yrs	0.207	0.405	0.256	0.436	0.297	0.457	0.289	0.453
Father Edu 13-15 yrs	0.258	0.437	0.236	0.424	0.215	0.411	0.154	0.361
Father Edu $\geq$ 16 yrs	0.282	0.450	0.161	0.368	0.276	0.447	0.169	0.375
Father Edu missing	0.129	0.335	0.180	0.384	0.049	0.217	0.069	0.253
Kessner Index= 2	0.267	0.442	0.320	0.467	0.251	0.434	0.299	0.458
Kessner Index= 3	0.057	0.232	0.116	0.320	0.076	0.265	0.179	0.384
Kessner Index missing	0.105	0.306	0.081	0.272	0.070	0.255	0.073	0.260
Private Insurance	0.519	0.500	0.379	0.485	0.592	0.492	0.388	0.487
Medicaid	0.347	0.476	0.481	0.500	0.225	0.418	0.320	0.467
Payment missing	0.035	0.184	0.026	0.159	0.083	0.276	0.092	0.289
WIC	0.349	0.477	0.482	0.500	0.319	0.466	0.397	0.489
WIC missing	0.112	0.315	0.097	0.295	0.021	0.144	0.032	0.176
Other Preg Outcomes	0.450	0.914	0.470	0.957	0.456	0.871	0.434	0.890
Any Med Risk Factor	0.375	0.484	0.380	0.485	0.259	0.438	0.222	0.416
Observations (Birth)	61892	61892	3333	3333	95268	95268	6609	6609
Number of Mothers	30946	30946	1111	1111	47634	47634	2203	2203

Table II: Characteristics of Nonsmokers and Smokers (Mothers with 2 Births, Gestation≥30)

Variable	Mothers at WA					Mothers at PA				
	Nonsmokers	Cessation before Preg	Cessation in the 1^{st} Tri	Cessation in the 2^{nd} Tri	Smoking through-out	Nonsmokers	Cessation before Preg	Cessation in the 1^{st} Tri	Cessation in the 2^{nd} Tri	Smoking through-out
Infant male	0.514	0.501	0.529	0.500	0.523	0.514	0.500	0.539	0.514	0.507
Parity	1.253	0.923	0.982	1.154	1.502	1.305	0.833	0.924	1.114	1.427
Mother's age	27.938	24.651	23.662	23.363	24.007	28.161	25.665	23.987	23.494	24.417
Mother non Hisp White	0.723	0.824	0.825	0.885	0.859	0.844	0.878	0.850	0.810	0.856
Mother non Hisp Black	0.033	0.025	0.040	0.021	0.034	0.110	0.078	0.118	0.150	0.109
Mother Edu = 12 yrs	0.215	0.320	0.352	0.410	0.377	0.222	0.360	0.414	0.442	0.450
Mother Edu 13-15 yrs	0.303	0.397	0.334	0.261	0.210	0.247	0.337	0.295	0.207	0.188
Mother Edu ≥ 16 yrs	0.320	0.093	0.043	0.017	0.017	0.379	0.179	0.083	0.041	0.029
Mother Edu missing	0.012	0.004	0.003	0.000	0.007	0.003	0.001	0.001	0.001	0.004
Mother Married	0.797	0.590	0.470	0.397	0.367	0.788	0.581	0.403	0.311	0.337
Father's Age	30.558	27.694	27.333	26.990	28.025	30.592	28.553	27.676	27.492	28.298
Father's Age missing	0.052	0.100	0.182	0.209	0.221	0.046	0.063	0.129	0.172	0.152
Private Insurance	0.554	0.407	0.314	0.248	0.211	0.645	0.615	0.463	0.353	0.309
Medicaid	0.316	0.357	0.486	0.590	0.641	0.161	0.296	0.401	0.528	0.525
WIC	0.314	0.469	0.581	0.688	0.647	0.248	0.414	0.543	0.625	0.640
Observations (Birth)	54781	1075	628	234	5174	74926	4442	2016	798	13086

Note: "WA" ("PA") represents the state of Washington (Pennsylvania).

Table III: Smoking Cessation and Infant Health(Mothers with 2 Births in 03-06, Gestation≥30)

	Dependent Variable: Birth Weight					
	WA		PA		Pooled	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	5.204	2.241	-1.110	-5.650	-0.939	-2.089
	(16.075)	(18.583)	(8.360)	(9.912)	(7.407)	(8.692)
Cessation in the 1^{st} Trimester(SK_1)	-9.330	-21.819	-24.382	0.788	-22.859	-2.360
	(20.893)	(23.650)	(12.544)*	(14.429)	(10.744)**	(12.225)
Cessation in the 2^{nd} Trimester(SK_2)	-99.874	-63.536	-137.310	-72.490	-132.861	-67.504
	(38.832)***	(38.099)*	(21.255)***	(21.622)***	(18.669)***	(18.699)***
Smoking throughout(SK_3)	-189.605	-69.036	-235.737	-103.666	-226.527	-89.841
	(9.028)***	(13.739)***	(6.236)***	(11.810)***	(5.101)***	(8.951)***
R^2	0.07	0.75	0.10	0.76	0.09	0.76
	Dependent Variable: Low Birth Weight					
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	-0.005	-0.017	0.002	-0.001	0.001	-0.004
	(0.006)	(0.008)**	(0.003)	(0.005)	(0.003)	(0.004)
Cessation in the 1^{st} Trimester(SK_1)	0.003	0.012	0.007	0.003	0.007	0.006
	(0.008)	(0.010)	(0.005)	(0.007)	(0.004)	(0.006)
Cessation in the 2^{nd} Trimester(SK_2)	0.040	0.027	0.038	0.007	0.040	0.012
	(0.017)**	(0.021)	(0.010)***	(0.013)	(0.009)***	(0.011)
Smoking throughout(SK_3)	0.035	0.021	0.044	0.016	0.043	0.018
	(0.004)***	(0.008)***	(0.003)***	(0.007)**	(0.002)***	(0.005)***
R^2	0.02	0.58	0.03	0.60	0.02	0.59
Observations (Births)	61892	61892	95268	95268	157160	157160
Number of Mothers	30946	30946	47634	47634	78580	78580

Note:

1. Additional controls include infant male, parity, infant birth year/month effects, mother's age, mother's race, mother's education, marital status, father's age/missing, father's race, father's education, number of other pregnant outcomes, any medical risk factor, Kessner index/missing, indicators of delivery payment types/missing, WIC participation/missing, residence at Pennsylvania. The birth invariant variables such as parental race are dropped in the mother fixed effects estimation.

2. Robust standard errors clustered at the mother's level in parentheses. *significant at 10%; ** significant at 5%; *** significant at 1%.

Table IV: Smoking Cessation and Infant Health(Mothers with 2 or 3 Births in 03-06, Gestation≥30)

	Dependent Variable: Birth Weight					
	WA		PA		Pooled	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	2.518	8.154	-0.996	-1.296	-1.562	3.521
	(15.989)	(18.252)	(8.156)	(9.583)	(7.253)	(8.439)
Cessation in the 1^{st} Trimester(SK_1)	-10.511	-14.806	-25.052	2.039	-23.856	1.434
	(20.493)	(22.650)	(12.151)**	(13.834)	(10.440)**	(11.736)
Cessation in the 2^{nd} Trimester(SK_2)	-107.912	-65.526	-137.304	-86.126	-134.789	-76.877
	(36.962)***	(36.503)*	(20.500)***	(20.986)***	(17.942)***	(18.140)***
Smoking throughout(Sk3)	-187.475	-58.502	-236.327	-105.357	-226.725	-86.579
	(8.782)***	(13.442)***	(6.023)***	(11.115)***	(4.938)***	(8.563)***
R^2	0.07	0.74	0.10	0.75	0.09	0.75
	Dependent Variable: Low Birth Weight					
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	-0.003	-0.012	0.002	-0.001	0.002	-0.003
	(0.006)	(0.008)	(0.003)	(0.005)	(0.003)	(0.004)
Cessation in the 1^{st} Trimester(SK_1)	0.002	0.008	0.007	0.004	0.007	0.005
	(0.008)	(0.009)	(0.005)	(0.007)	(0.004)	(0.006)
Cessation in the 2^{nd} Trimester(SK_2)	0.041	0.027	0.036	0.010	0.039	0.014
	(0.017)***	(0.020)	(0.010)***	(0.013)	(0.008)***	(0.011)
Smoking throughout(SK_3)	0.034	0.019	0.045	0.018	0.043	0.018
	(0.004)***	(0.008)**	(0.003)***	(0.007)***	(0.002)***	(0.005)***
R^2	0.01	0.57	0.03	0.59	0.02	0.58
Observations (Births)	65225	65225	101877	101877	167102	167102
Number of Mothers	32057	32057	49837	49837	81894	81894

Note: the same as Table III.

Table V: Smoking Cessation and Infant Health(Mothers with 2 Births in 03-06, Gestation≥28)

	Dependent Variable: Birth Weight					
	WA		PA		Pooled	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	3.265	0.077	-3.178	-7.448	-3.193	-3.917
	(16.334)	(19.044)	(8.506)	(10.123)	(7.535)	(8.880)
Cessation in the 1^{st} Trimester(SK_1)	-11.101	-23.312	-26.862	-3.802	-25.452	-6.113
	(21.328)	(24.233)	(12.758)**	(14.765)	(10.937)**	(12.516)
Cessation in the 2^{nd} Trimester(SK_2)	-115.077	-69.576	-171.674	-108.306	-163.378	-96.773
	(40.228)***	(39.384)*	(23.030)***	(23.622)***	(20.058)***	(20.244)***
Smoking throughout(SK_3)	-191.737	-70.806	-236.671	-101.953	-228.058	-89.53
	(9.148)***	(14.105)***	(6.316)***	(12.073)***	(5.167)***	(9.167)***
R^2	0.07	0.75	0.10	0.76	0.09	0.75
	Dependent Variable: Low Birth Weight					
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	-0.004	-0.015	0.003	-0.000	0.002	-0.003
	(0.006)	(0.008)	(0.003)	(0.005)	(0.003)	(0.004)
Cessation in the 1^{st} Trimester(SK_1)	0.004	0.014	0.008	0.006	0.008	0.009
	(0.008)	(0.010)	(0.005)	(0.008)	(0.005)*	(0.006)
Cessation in the 2^{nd} Trimester(SK_2)	0.046	0.028	0.052	0.024	0.052	0.025
	(0.018)***	(0.021)	(0.011)***	(0.014)*	(0.009)***	(0.012)**
Smoking throughout(SK_3)	0.036	0.023	0.045	0.017	0.044	0.019
	(0.004)***	(0.008)***	(0.003)***	(0.008)**	(0.003)***	(0.006)**
R^2	0.02	0.59	0.03	0.60	0.02	0.60
Observations (Births)	62070	62070	95600	95600	157670	157670
Number of Mothers	31035	31035	47800	47800	78835	78835

Note: the same as Table III.

Table VI: Smoking Cessation and Infant Health(Mothers with 2 Births in 03-06, Gestation≥28, Control for Gestation)

	Dependent Variable: Birth Weight					
	WA		PA		Pooled	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	7.038	-7.084	-2.840	-12.072	-0.706	-9.348
	(14.223)	(15.905)	(7.050)	(8.403)	(6.304)	(7.394)
Cessation in the 1^{st} Trimester(SK_1)	4.432	-9.890	-22.021	-5.021	-16.606	-4.121
	(17.532)	(20.137)	(10.674)**	(12.120)	(9.117)*	(10.303)
Cessation in the 2^{nd} Trimester(SK_2)	-67.979	-55.082	-93.858	-59.433	-89.798	-55.916
	(29.256)**	(31.260)*	(15.573)***	(17.603)***	(13.756)***	(15.251)***
Smoking throughout(SK_3)	-152.464	-63.587	-198.316	-89.657	-186.924	-79.391
	(7.635)***	(11.748)***	(5.130)***	(9.827)***	(4.233)***	(7.533)***
Gestation	169.420	147.405	171.001	148.229	170.483	147.919
	(1.462)***	(1.868)***	(0.984)***	(1.300)***	(0.824)***	(1.072)***
R^2	0.32	0.81	0.36	0.83	0.35	0.82
	Dependent Variable: Low Birth Weight					
Mother Fixed Effects?	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	-0.005	-0.013	0.003	0.002	0.001	-0.001
	(0.005)	(0.008)*	(0.002)	(0.005)	(0.002)	(0.004)
Cessation in the 1^{st} Trimester(SK_1)	-0.000	0.009	0.007	0.006	0.005	0.008
	(0.007)	(0.009)	(0.005)	(0.007)	(0.004)	(0.005)
Cessation in the 2^{nd} Trimester(SK_2)	0.031	0.022	0.023	0.004	0.027	0.009
	(0.014)**	(0.019)	(0.008)***	(0.012)	(0.007)***	(0.010)
Smoking throughout(SK_3)	0.024	0.020	0.031	0.011	0.030	0.015
	(0.004)***	(0.007)***	(0.003)***	(0.007)*	(0.002)***	(0.005)***
Gestation	-0.053	-0.053	-0.062	-0.061	-0.059	-0.058
	(0.001)***	(0.001)***	(0.001)***	(0.001)***	(0.001)***	(0.001)***
R^2	0.21	0.65	0.25	0.67	0.23	0.67
Observations (Births)	62070	62070	95600	95600	157670	157670
Number of Mothers	31035	31035	47800	47800	78835	78835

Note: the same as Table III.

Table VII: Results for Newly Defined Smokers and Smoking Intensity(Mothers with 2 Births in 03-06, Gestation≥30)

	Dependent Variable: Birth Weight				Dependent Variable: Low Birth Weight			
	Newly Defined Smokers		Smoking Intensity		Newly Defined Smokers		Smoking Intensity	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother Fixed Effects?	N	Y	N	Y	N	Y	N	Y
Cessation before Pregnancy(SK_0)	-0.861	-0.826	-1.166	-1.805	0.001	-0.004	0.001	-0.004
	(7.310)	(8.557)	(7.407)	(8.692)	(0.003)	(0.004)	(0.003)	(0.004)
Cessation in the 1^{st} Trimester(SK_1)	-23.199	-3.620	-23.083	-2.089	0.007	0.009	0.007	0.006
	(10.380)**	(11.859)	(10.743)**	(12.226)	(0.004)*	(0.006)	(0.004)*	(0.006)
Cessation in the 2^{nd} Trimester(SK_2)	-133.053	-72.078	-114.154	-87.809	0.041	0.018	0.032	0.016
	(17.643)***	(17.721)***	(23.163)***	(23.699)***	(0.008)***	(0.011)*	(0.011)***	(0.014)
Smoking throughout(SK_3)	-218.507	-84.727	-192.992	-79.583	0.042	0.019	0.040	0.021
	(4.944)***	(8.523)***	(7.137)***	(10.066)***	(0.002)***	(0.005)***	(0.004)***	(0.006)***
SK_2×(avg cigs≥10, 1^{st} and 2^{nd} Trimester)			-49.546	52.888			0.022	-0.011
			(37.627)	(38.069)			(0.018)	(0.024)
SK_3×(avg cigs≥10, 1^{st} and 2^{nd} Trimester)			-53.810	-20.371			0.005	-0.005
			(8.220)***	(9.588)**			(0.004)	(0.006)
R^2	0.09	0.76	0.09	0.76	0.02	0.59	0.02	0.59
Observations (Births)	159298	159298	157160	157160	159298	159298	157160	157160
Number of Mothers	79649	79649	78580	78580	79649	79649	78580	78580

Note: the same as Table III.

Table VIII: Smoking Cessation and "Prenatal Smokers"(Mothers with 2 Births in 03-06, Any Gestation)

	Dependent Variable: Birth Weight	
	(1)	(2)
Mother Fixed Effects?	N	Y
Prenatal Smokers(Smoking through 2^{nd} or 3^{rd} Trimester)	-228.634	-92.552
	(5.234)***	(9.379)***
R^2	0.09	0.74
Prenatal Smokers(Smoking in every Trimester)	-221.872	-62.851
	(5.238)***	(9.734)***
R^2	0.08	0.74
Prenatal Smokers(Smoking in any Trimester)	-204.776	-70.976
	(5.011)***	(12.226)***
R^2	0.08	0.74
	Dependent Variable: Low Birth Weight	
Mother Fixed Effects?	N	Y
Prenatal Smokers(Smoking through 2^{nd} or 3^{rd} Trimester)	0.046	0.021
	(0.002)***	(0.005)***
R^2	0.03	0.60
Prenatal Smokers(Smoking in every Trimester)	0.042	0.010
	(0.003)***	(0.005)**
R^2	0.03	0.60
Prenatal Smokers(Smoking in any Trimester)	0.042	0.020
	(0.002)***	(0.007)***
R^2	0.03	0.60
Observations (Births)	162366	162366
Number of Mothers	81183	81183

Note: the same as Table III.

Appendix

Data Construction

The Washington State Longitudinal Births Database was originally constructed jointly by the Washington State Department of Health and the Department of Epidemiology at the University of Washington. According to their instruction, linking multiple births of the same mother is achieved through a two-step procedure. In step one, the mother's maiden name and first name are used to identify the potential "same mother" across births. In step two, the "same mothers" are refined by comparing other matching variables. The crucial process is to compare all the birth records per "same mother" in six combinations of the seven matching variables including mother's date of birth, date of the last live birth, parity, race, state of birth, middle initial, and resident address. In particular, every combination uses mother's birth date while only one combination applies her resident address, with the remaining matching variables utilized in about three to five combinations. The mothers identified as inconsistent matching in this step are excluded. I place three more restrictions to get the final Washington State mother sample for this paper. First, I drop all the mothers who were not Washington State residents. Second, only the mothers with two or three singleton births are kept. Third, all the matched births to each mother must be consistently sequential.

A very similar matching strategy is used to construct the Pennsylvania State panel data of sibling births. Under special permission from the Pennsylvania Department of Health, I have acquired access to a restricted version of this state's Natality Birth data from 2003 to 2006 which contains mother's name, mother's date of birth, and infant's date of birth. Then, mothers are included in the Pennsylvania panel data sample if five criteria are met. First, I only keep the mothers with exact matching on their maiden names, first names, and dates of birth. Second, the mothers whose self-reported races are inconsistent across births are excluded. Third, I require the linked births to be consistently sequential by checking a match on either the parity or the birth date of the last live birth. Fourth, I drop all the mothers who were not Pennsylvania State residents[30]. Fifth, only mothers who delivered two or three singleton births during that period are kept.

[30]Mother's state of birth is not used as a matching variable because it is not available.

Table AI: The Adoption of 2003 U.S. Standard Certificates of Live Birth across 50 States)

States using 2003 certificates by 2006	2003	2004	2005	2006	Mothers giving births in 2003
DE	N	N	N	Y	11329
ID	N	Y	Y	Y	21800
KS	N	N	Y	Y	39476
KY	N	Y	Y	Y	55236
NE	N	N	Y	Y	33647
NH	N	N	Y	Y	14393
NY(excluding New York City)	N	Y	Y	Y	133532
ND	N	N	N	Y	7972
OH	N	N	N	Y	149679
PA	**Y**	**Y**	**Y**	**Y**	**145959**
SC	N	N	Y	Y	55649
SD	N	Y	N	Y	11027
TN	N	Y	Y	Y	78890
TX	N	N	Y	Y	377476
VT	N	N	N	Y	6589
WA	**Y**	**Y**	**Y**	**Y**	**80439**
WY	N	N	N	Y	6700

Source: National Natality Data on Live Birth 2003, 2004, 2005 and 2006

Table AII: Patterns of Maternal Smoking Before and During Pregnancy

Smoking prior to and during pregnancy				Types of Smokers		Mothers at WA		Mothers at PA	
Before pregnancy?	1^{st} trimester?	2^{nd} trimester?	3^{rd} trimester?	Definition 1	Definition 2	2 Births	3 Births	2 Births	3 Births
Y	Y	Y	Y	Sk3	Sk3	5260	450	13438	1278
Y	Y	Y	N	Sk2	Sk2	241	20	835	57
Y	Y	N	N	Sk1	Sk1	640	37	2087	119
Y	N	N	N	Sk0	Sk0	1092	45	4550	223
N	N	N	N	Nonsmokers	Nonsmokers	54846	2808	75114	5052
Y	Y	N	Y	Excluded	Sk3	42	4	196	14
Y	N	Y	Y	Excluded	Sk3	59	4	245	18
N	Y	Y	Y	Excluded	Sk3	16	1	38	4
Y	N	N	Y	Excluded	Sk3	28	4	210	14
N	N	N	Y	Excluded	Sk3	30	1	119	15
N	Y	N	Y	Excluded	Sk3	0	0	6	1
N	N	Y	Y	Excluded	Sk3	10	0	31	1
Y	N	Y	N	Excluded	Sk2	8	0	34	0
N	Y	Y	N	Excluded	Sk2	5	0	3	2
N	N	Y	N	Excluded	Sk2	7	1	18	2
N	Y	N	N	Excluded	Sk1	26	0	64	1

Note: Sk0 indicates "cessation before pregnancy", Sk1 represents "cessation in the 1^{st} trimester", Sk2 indicates "cessation in the 2^{nd} trimester" and Sk3 represents "smoking throughout".

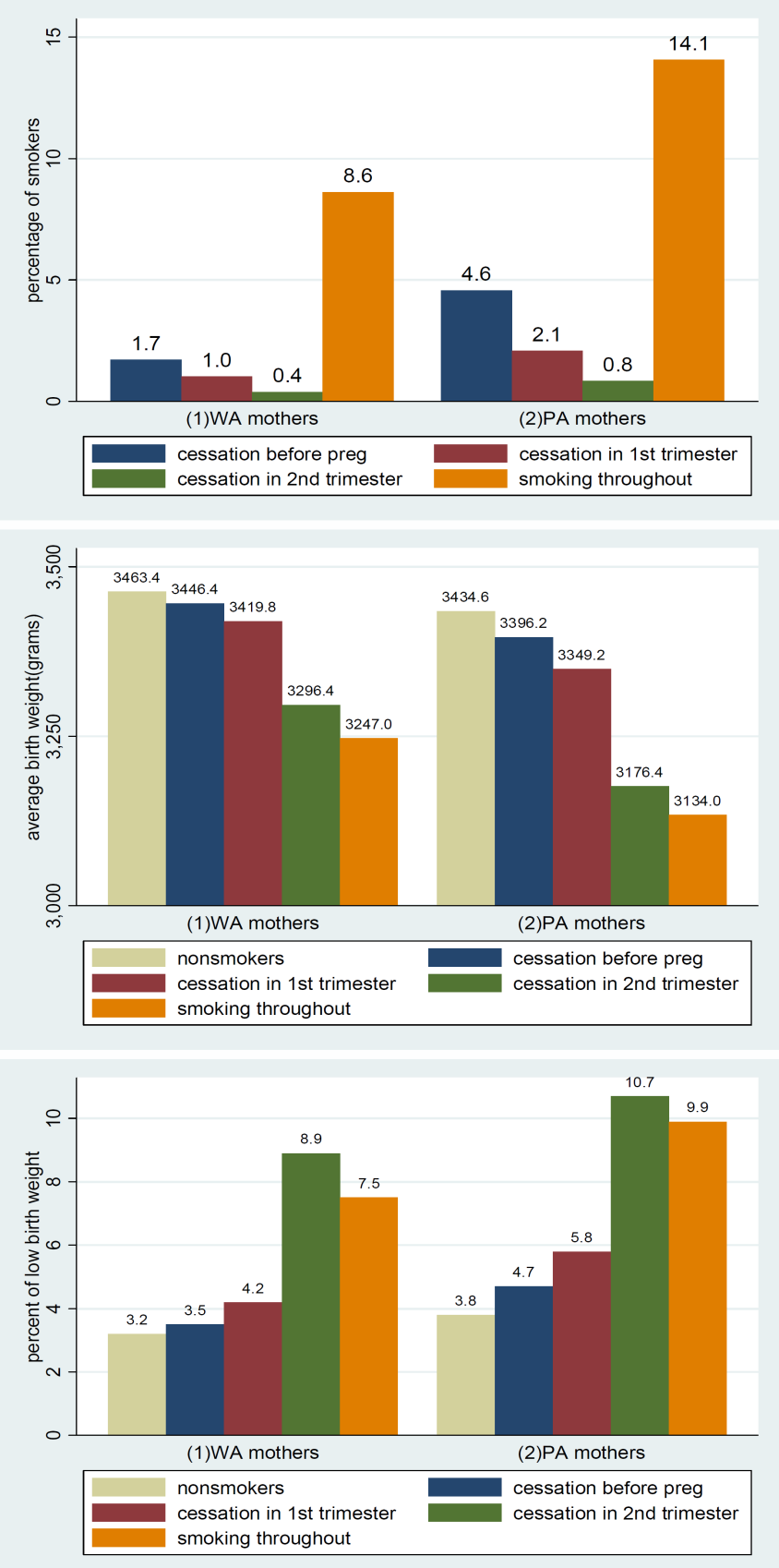

Figure A1: Smoking Cessation and Infant Health (Mothers with 2 or 3 Births, Gestation ≥ 28)